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Effect of *Escherichia coli* Lipopolysaccharide on Human Platelets

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ABSTRACT

In this study, *Escherichia coli* lipopolysaccharide (LPS) dose-dependently (100-500 μg/mL) and time-dependently (10-60 min) inhibited platelet aggregation in human platelets stimulated by agonists. In lactate dehydrogenase (LDH) assay, LPS (100-500 μg/mL) induced a slight release (7% ~ 9%) of LDH from cell cytosol in human platelets within a 10-min incubation period. LPS (200 μg/mL) added to human platelet suspensions for the indicated times (10-60 min) also induced a slight increase in LDH. At the same concentration (100-500 μg/mL), the extent of LPS used to trigger LDH release was lower than that needed for inhibition of platelet aggregation. On the other hand, to examine the apoptotic activity of LPS, human leukemia cells (HL-60) were used instead of platelets in this study. We found that LPS (10 and 100 μg/mL) did not significantly influence propidium iodide (PI) binding to DNA in HL-60 cells with a 5-day incubation period. These results indicate that the antiplatelet activity of *Escherichia coli* LPS might not result from cytolytic activities.

INTRODUCTION

Endotoxins, high-molecular-weight complexes of lipopolysaccharides (LPS), are major components of the outer membranes of the cell walls of gram-negative bacteria which are shed from bacteria when cell lysis occurs and to a lesser extent during active growth.[1] Endotoxin has a broad variety of biological effects, and when injected locally, an intense inflammatory reaction occurs, which is characterized by increased vascular permeability, hyperemia, microthrombosis, and leucocyte infiltration.[2] *Escherichia coli* LPS has also been shown to cause the clinical syndrome known as septic shock.[3] In addition, clinical and experimental endotoxemia is often associated with intravascular coagulation, such as renal cortical necrosis.[4] Tissue damage subsequently results not only from reduced perfusion of the exchange vessels,

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